CASE REPORTS

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Lobar Consolidation and Hemorrhagic Bronchitis in Chronic Eosinophilic Pneumonia

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SINCE ITS INITIAL DESCRIPTION, chronic eosinophilic pneumonia has been defined as a disease of lung parenchyma that is recognized by a characteristic clinical history and a roentgenographic pattern of scattered nonsegmental peripheral infiltrates.[1-4] It has been emphasized recently that these features are sufficiently specific to allow definitive diagnosis without the necessity of lung biopsy in most instances.[3,4] In this report we describe a patient with combined eosinophilic pneumonia and bronchitis whose atypical presentation resulted in diagnostic confusion and delay of appropriate therapy.

Report of a Case

A 46-year-old woman who did not smoke was admitted with complaints of progressive dyspnea and hemoptysis. Her past medical history was notable only for menorrhagia secondary to biopsy-proved chronic endometritis. Six weeks before we saw her, a persistent nonproductive cough, anorexia and fatigue had developed. Two weeks before admission to our facility, she was admitted to another hospital with complaints of hectic fever, night sweats, mild dyspnea and weight loss of 7 kg. She had no history of asthma, atopy or the use of any medicine. A chest radiograph (Figure 1) showed right upper lobe consolidation and was interpreted as compatible with lobar pneumonia. Stain and culture of the sputum did not identify bacterial, fungal or mycobacterial pathogens. The total leukocyte count was 11,500 per cu mm with 5 percent eosinophils, and the hemoglobin value was 9.6 grams per dl. Erythromycin therapy (250 mg four times a day) was begun, but the patient experienced little subjective improvement. No rectal temperature exceeding 38.9°C (102°F) was recorded, and she was discharged on the third hospital day with presumptive diagnoses of bacterial or mycoplasmal pneumonia and iron deficiency anemia caused by menstrual blood loss.

Ten days later she was admitted to our hospital because of progression of symptoms despite antibiotic therapy. She appeared cachectic, diaphoretic and dyspneic at rest. Her vital signs were oral temperature 39.4°C (102.9°F), respirations 32 per minute, pulse rate 96 per minute, and blood pressure 100/60 mm of mercury. Tubular breath sounds were heard bilaterally over the upper lung zones, and no wheezes were detected. The remainder of the physical examination was unremarkable.

The chest roentgenogram (Figure 2) showed extension of the original infiltrate to involve the peripheral regions of the upper and middle lung zones, bilaterally, with sparing of central areas. Abnormal laboratory values included: hemoglobin 8.1 grams per dl, leukocytes 15,500 per cu mm with an absolute eosinophil count of 910 per cu mm, albumin 2.1 grams per dl and erythrocyte sedimentation rate (Westergren) 95 mm per hour. Blood film and erythrocyte indices indicated hypochromia and microcytosis. The test for serum creatinine was 1.0 mg per dl, and analysis of urine was unremarkable. Sputum was blood-tinged and contained numerous segmented cells but scant flora; a Wright stain for eosinophils was not done. Skin tests with tuberculin, mumps and *Candida* antigens were nonreactive. Stool and sputum examinations for ova and parasites and serum tests for antinuclear antibodies and rheumatoid factor were negative. Assay for total hemolytic complement ($C'H_{50}$) was within expected normal values.

The patient's total lung capacity and single breath diffusing capacity for carbon monoxide were 34 percent and 48 percent of the predicted

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values, respectively. No airflow obstruction was detected using a spirometer. Arterial blood gas values while breathing room air were: pH 7.50, Po_2 59 torr, and Pco_2 34 torr.

Because of the patient's radiographic and clinical features, the differential diagnoses considered were tuberculosis, partially treated bacterial pneumonitis and eosinophilic pneumonia. Following discontinuation of antibiotic drugs, blood and sputum cultures were obtained, antituberculous therapy was begun and three units of packed erythrocytes were given. Because her diagnosis continued to be uncertain, fiberoptic bronchoscopy was carried out on the fourth hospital day. All passages were widely patent to the subsegmental level, but diffuse hemorrhagic bronchitis involved both central and peripheral airways. Multiple endobronchial biopsy specimens were obtained from the right main bronchus and transbronchial specimens were obtained from the right upper lobe.

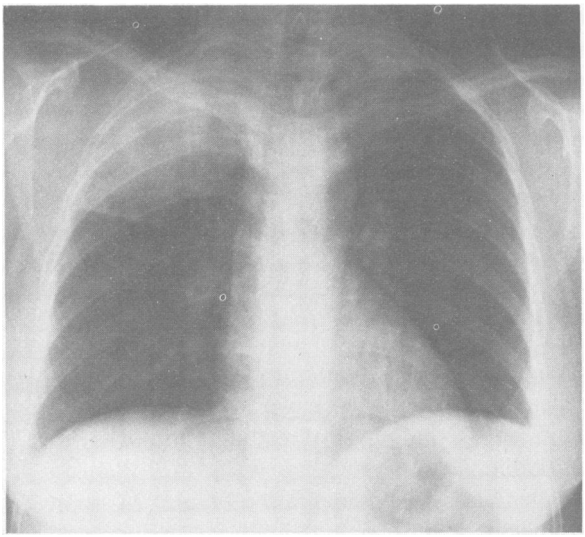

Figure 1.—Initial chest roentgenogram, showing consolidation of the right upper lobe.

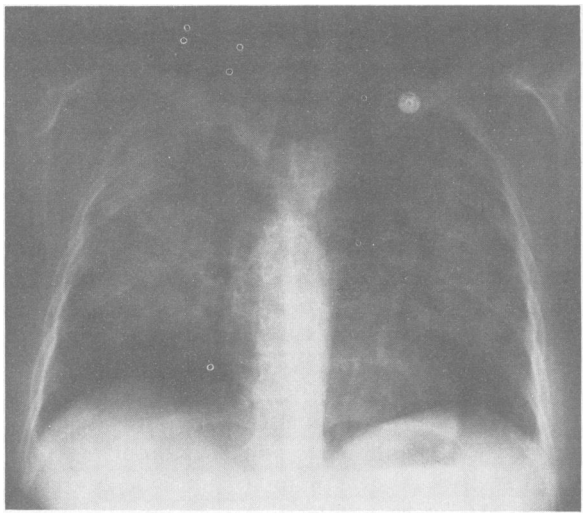

Figure 2.—Chest roentgenogram taken during second hospital stay, showing widespread peripheral infiltrates with central zone sparing characteristic of chronic eosinophilic pneumonia.

Histopathologic findings were those described for chronic eosinophilic pneumonia.[1,2] The biopsy specimen of pulmonary parenchyma (Figure 3A) showed alveolar and interstitial filling with histiocytes, lymphocytes, fibroblasts and eosinophils. Scattered neutrophils and giant cells were identified. The bronchial mucosa (Figure 3B) was thickened by a similar infiltrate of eosinophils and histiocytes. No bacterial, fungal or acid-fast organisms were detected when the mucosa was stained or cultured. Acute and convalescent titers for cold agglutinins and serum antibodies to mycoplasma pneumoniae, legionella pneumophila, influenza A and B, adenovirus, chlamydia and

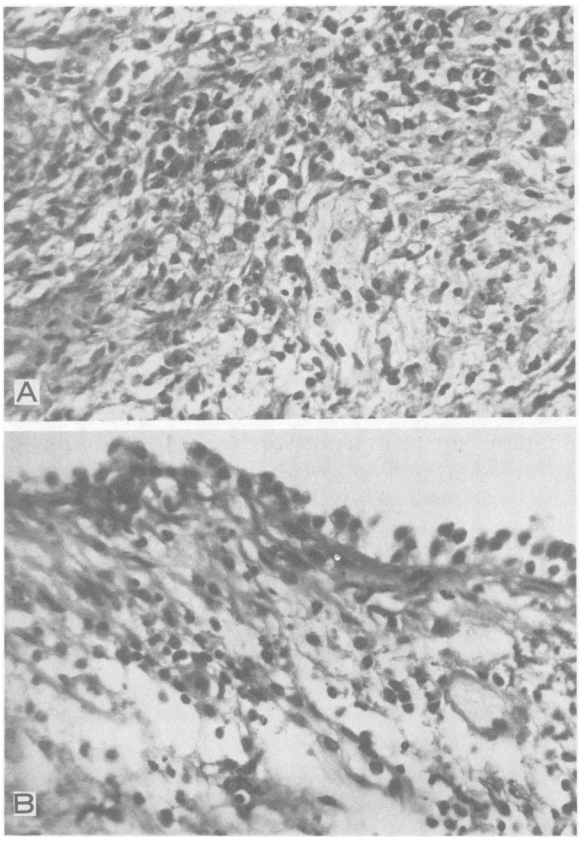

Figure 3.—Biopsy specimens of pulmonary parenchyma (upper panel, A) and of mucosa from a central bronchus (lower panel, B). An infiltrate of eosinophils and hisitocytes is present in both specimens. (Hematoxylin-eosin stain, original magnification ×400.)

respiratory syncytial virus were all less than 1:16. No rise in titer between samples was observed for any organism.

Therapy with 60 mg of prednisone per day was begun, and 36 hours later the patient's constitutional symptoms had improved substantially. Dyspnea rapidly diminished, and there were no further episodes of hemoptysis. A chest roentgenogram showed convincing clearing by the sixth day of corticosteroid treatment. The patient was discharged to her home on the 14th hospital day to continue prednisone and iron sulfate therapy. Subsequently, findings on a chest roentgenogram as well as erythrocyte sedimentation rate, hemoglobin and eosinophil counts returned to normal; all pulmonary function abnormalities reversed as well. Three months following discharge, the patient was still asymptomatic on low-dose alternate-day corticosteroid therapy.

Discussion

Previous descriptions of chronic eosinophilic pneumonia have urged recognition of a distinctive radiographic and clinical entity.[1,3,5] The present case illustrates that this disease may also present atypically with isolated lobar consolidation and hemorrhagic bronchitis.

In the original report of Carrington and his associates[1] three radiographic features were observed uniformly: dense nonsegmental peripheral infiltrates with sparing of central zones ("photographic negative of pulmonary edema"), rapid resolution on corticosteroid therapy and recurrence of the lesions in the same location during relapse. These points have been supported in more current literature by the observations of numerous authors.[2-11] In separate reviews, Dines,[5] Angelillo,[10] Gaensler[3] and Leitch[12] and their colleagues recently called attention to the importance of the characteristic chest roentgenogram, and on this basis cogently argued that the diagnosis is secure without tissue documentation in the great majority of cases in which the features are stereotypic. It is also important to emphasize that atypical presentations occur; radiographs that do not conform to the standard pattern have been described.[3] However, a roughly segmental distribution has been reported in only two previous instances,[11,13] and isolated lobar consolidation, as observed initially in our patient, has not been described previously.

Unlike other diseases of pulmonary eosinophilia, there is an almost complete lack of histological information concerning the conducting airways in chronic eosinophilic pneumonia. Cough and sputum production are frequent complaints, but to our knowledge the present case is the first in which diffuse eosinophilic bronchitis has been documented. Similarly, hemoptysis is recognized to occur in this disease, but little attention has been directed toward defining its site of origin. In their pathological description of eosinophilic pneumonia, Liebow and Carrington[2] showed hemosiderin-laden alveolar macrophages in a single instance in which hemoptysis occurred, and a parenchymal bleeding source was implied. Our observations in this patient indicate that the inflammatory process occasionally extends to involve the proximal airways, and that hemorrhage is a potential consequence of that involvement.

Despite its extraordinary response to appropriate therapy, eosinophilic pneumonia is potentially life threatening if unrecognized. While usually identifiable by a distinctive presentation, this disease is not confined to a unique syndrome and deserves diagnostic consideration even in cases in which highly atypical features are seen.

Summary

Isolated lobar infiltrates and hemoptysis characterized the condition of a 46-year-old woman with a subacute illness. Based on subsequent radiographs and parenchymal biopsy specimens, the diagnosis of chronic eosinophilic pneumonia was established. Despite its usual description as a parenchymal disease with a distinctive roentgenographic pattern, the atypical features of endobronchial involvement and segmental consolidation seen in this patient are consistent with this diagnosis.

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